Journal of Orthopaedic Science and Research

ATHENAEUM SCIENTIFIC PUBLISHERS

Research Article

Incidence of Midfoot Instability in Patients with Achilles Tendon Ruptures (ATR): Why ATR is a Secondary Sign of Spring Ligament Laxity

Luisa Valentini[1], Md Abdul Aziz[1*], Simrin Chaudhri[1], Steven O Famure[1], Zhikai Li[2], Chandra Pasapula[1*]

[1]The Queen Elizabeth Hospital Kings Lynn NHS Foundation Trust, United Kingdom

[2]Corpus Christi College, University of Cambridge, United Kingdom

***Correspondence author:** Md Abdul Aziz, Clinical Fellow Trauma and Orthopaedic, The Queen Elizabeth Hospital, King's Lynn NHS Foundation Trust Gayton Rd, King's Lynn, United Kingdom, PE30 4ET and Chandra Pasapula, T and O Consultant, The Queen Elizabeth Hospital, King's Lynn NHS Foundation Trust, Gayton Rd, King's Lynn, United Kingdom, PE30 4ET; Email: md.aziz@qehkl.nhs.uk; ChandraS.Pasapula@qehkl.nhs.uk

Citation: Valentini L, et al. Incidence of Midfoot Instability in Patients with Achilles Tendon Ruptures (ATR): Why ATR is a Secondary Sign of Spring Ligament Laxity. J Ortho Sci Res. 2025;6(1):1-12. https://doi.org/10.46889/JOSR.2025.6105

Received Date: 14-01-2025

Accepted Date: 27-01-2025

Published Date: 06-02-2025

Abstract

Achilles Tendon Rupture (ATR) is a common yet debilitating injury that affects individuals of all ages and activity levels. Several theories describe the pathogenesis of ATR. This study aims to evaluate if there is increased medial column instability in terms of talonavicular laxity or first ray instability in patients with ATR. Patients were recruited from a database of Tendoachilles (TA) ruptures presenting to the clinic. All patients underwent non operative treatment. Patients with pre-existing foot surgery, arthropathy or generalised laxity were excluded. A total of 14 TA ruptures were assessed for First Ray (FR) instability and Spring Ligament (SL) incompetence. Results: No patients had pre-existing Achilles problems or foot instability in our cohort. One patient had a chronic unilateral rupture, missed on initial presentation and had treatment. TA maximum Anteroposterior (AP) thickness between affected and unaffected feet demonstrated no significant difference. ATR feet demonstrated greater midfoot instability in terms of both Talonavicular (TN) laxity and First Ray Instability in all ATR feet ($p<0.05$). Despite medial column instability the incidence of significant valgus impingement pain was 8/14 cases. Both were statistically increased. This suggests that combined pathogenesis may be responsible for both pathologies. In conclusion, this is the first study to our knowledge that reports increased medial column laxity being present in all ATR feet. External biomechanical factors in a predisposed foot help generate an internal moment/ force that overloads the TA. The relationship between intrinsic foot biomechanics and ATR has not been described. Future treatments may therefore be directed at restoring midfoot stability using orthotics or surgery to help restore biomechanics and to help offload the TA and protect the foot from future re-ruptures.

Keywords: Achilles Tendon Rupture; Medial Column; Midfoot Instability; Spring Ligament Laxity; First Ray Instability; Foot Biomechanics

Introduction

Achilles Tendon Rupture (ATR) is a prevalent musculoskeletal injury, with an incidence ranging from 6 to 37 cases per 100,000 person-years [1]. Its occurrence spans diverse age groups and activity levels, with a noted increase among individuals over 45 years old [2-6]. A rising incidence is partly attributed to the rise in sports activities that involve running and jumping [7]. Despite the high mechanical demands of these activities, the incidence of ATR remains relatively low among athletes, emphasizing the multifactorial nature of its pathogenesis [7]. The consequences of ATR are devastating [8]. ATR often occurs in non-athletic settings and older populations, with devastating consequences for functional mobility. Professional athletes, for instance, face significant challenges, with 25% unable to return to their pre-injury performance levels [9].

Higher incidence with increased Body Mass Index (BMI) alludes to biomechanical causes. Oral quinolone/ corticosteroid use, age and race are not significant risk factors in ATR [10]. Interestingly, conditions such as Diabetes Mellitus (DM) are not associated with increased rupture risk, despite their impact on tendon stiffness through non-enzymatic glycosylation [11,12].

The true pathogenesis of overload causing rupture may arise internally within the foot, from an internally derived opposing moment generated secondary to midfoot failure. Existing literature has extensively examined external biomechanical contributors to ATR, but the role of intrinsic foot mechanics remains underexplored. This presence of medial column instability in ATR has not been evaluated to date.

Aim

To quantify medial column instability for both talonavicular laxity and first ray instability in patients with ATR.

Methods

A retrospective case-matched study was performed. Ethical approval was obtained for this study. Patients presenting to the clinic with an ATR between 5/7/2022 to 10/7/2023 were identified. The history was obtained to determine the mechanism of injury and foot position at the time of injury to help predict the biomechanical environment the foot was in at the time of rupture. Assessment for symptoms of instability, giving way, prior Achilles pain, diabetes and lipid disorders was undertaken. Tendinopathy due to the long-term statin, steroid and ciprofloxacin use was determined and excluded if present. Patients with seronegative arthropathies and inflammatory disorders such as gout were excluded.

Measuring First Ray Instability (FRI)

A custom-made digital scale foot Ankle Orthosis (AFO), quantified first ray dorsal sagittal instability. This was similar to Klaue's device, validated by Coughlin. To negate the effect of increased FRI with ankle plantar flexion, all measurements were taken in neutral ankle position. The middle column was held to the orthotic base by fixing the second metatarsal head manually, to resist middle column dorsiflexion during dorsal force application to the plantar metatarsal. A dorsal force was applied until a firm endpoint reached. A probe linked to a sliding caliper in contact to the dorsal metatarsal head enabled us to quantify dorsal metatarsal head translation. Dorsal readings negated the effect of fat pad compression that may distort plantar surface readings. A mean of three readings were taken and rounded to 0.1 mm. Translation greater than 8 mm was considered significant as proposed by Klaue/Glasoe as having pathological significance to define the presence of FRI.

Measuring SL Incompetence

SL strain was assessed by lateral foot translation score. Immobilisation of the calcaneus and talus in a padded clamp-controlled leg rotation and hindfoot eversion. Further, hindfoot external rotation instability from deep deltoid instability would be controlled with calcaneal and talus immobilisation. Subtalar joint immobilisation inhibited hindfoot pronation. This immobilisation and a lateral force applied to the medial first metatarsal head until a firm endpoint was reached, ensured lateral midfoot abduction force at the talonavicular joint with forefoot translation with no tibial rotation. The talonavicular axis and the first metatarsal length then visually amplifies SL strain that can then be quantified (see diagram). A mean of 3 metatarsal head lateral translation scores in mm was recorded. Validation for using 'lateral translation scores' comes from previous anatomic studies which indicated scores of less than 20 mm implied SL intact but increased to 40-50 mm (load dependent) on SL sectioning (Fig. 1) [13].

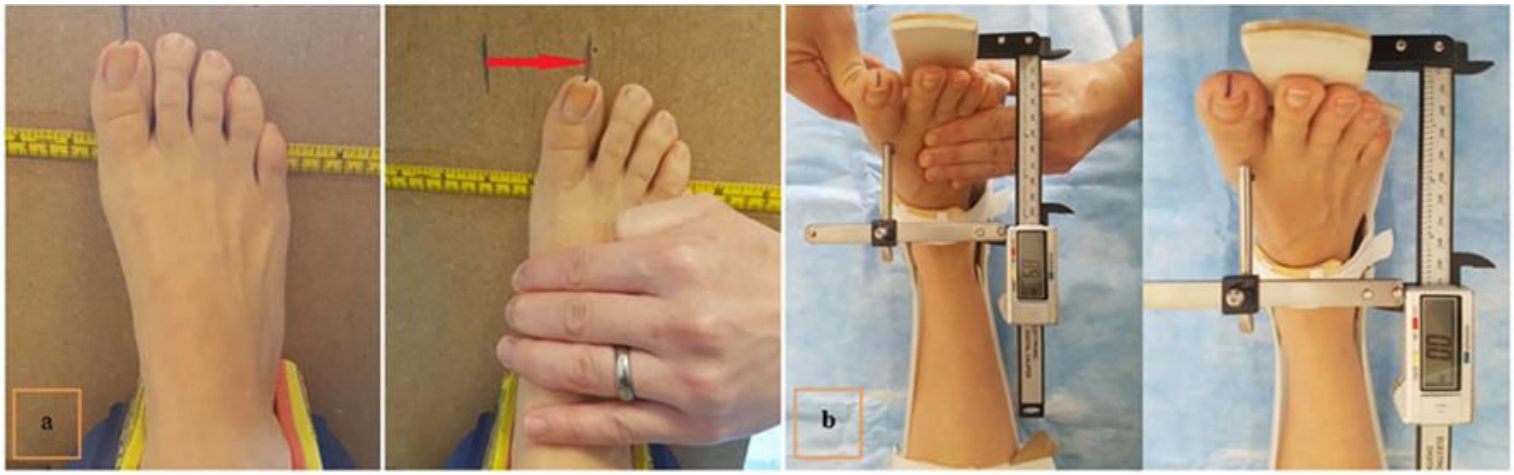

Figure 1: Demonstration of modified device used to measure (a) Lateral translation by fixing heel in the neutral position and measuring with firm pressure the lateral most distance of the forefoot and (b) Dorsal translation (or TMT instability) by measuring with firm pressure the dorsal most distance of the first TMT joint.

Age, sex, BMI, symptom duration, pain scores, treatment history, TA and gastrocnemius tightness were recorded measured with a goniometer with the foot in slight inversion. Beighton scores helped differentiate local laxity from generalised laxity. Average duration from rupture was calculated and duration in the boot was recorded. The position of the foot at the time of the rupture was identified if possible. Ruptures were also classified as contact and non-contact.

Tenderness was manually palpated for at the tibialis posterior and valgus impingement and maximum Visual Analogue Scale (VAS) scores were recorded. MRI sensitivity in quantifying SL strain is unknown [14]. US was used in our study to assess the SL for tears. US imaging measured TA maximal thickness and distance from the superior calcaneus to the most tender point of the TA. SL thickness was measured at the level of the talar neck, beneath the tibialis posterior tendon.

The Kolmogorov-Smirinov test was used to assess data for normality. A paired t test was used to assess parametric data. A p-value of less than 0.05 was taken as significant.

A power calculation determined sample size. An alpha value of 0.8 and beta value of 0.05 were used. Standard deviations were estimated from previous studies. For FRI a mean score of 3.5 mm (Standard Deviation (SD) of 3 and an 8 mm for unaffected and affected side respectively). A sample size of 14 was required. For lateral translation scores a mean of 25 mm (SD 9 mm) for unaffected feet with mean affected value of 55 mm required sample size of 4 patients. Intraclass coefficients were used to assess intra-observer reliability.

Results

A total of 26 patients were identified between 5/7/2022 to 10/7/2023 presenting with unilateral TA ruptures to the clinic. 4 were excluded due to high Beighton scores, seronegative disease or preexisting ankle fractures/trauma. Of the 22 patients invited, 14 patients came for assessment. No patients had pre-existing Achilles problems or foot instability. one patient had a chronic unilateral rupture which was missed at initial presentation. All other patients had non operative serial cast treatment, with sequential dorsiflexion over a period of 4-12 weeks with non-weight bearing. The mean Post-Operative (POP) immobilisation time was 8.21 weeks (for the acute) with cast changes and immobilisation (range 4 to 12 weeks). Patients did not have early weight bearing or early range of motion. 10 patients were transitioned into a full weight bearing boot after POP immobilisation. All patients received physiotherapy once their POP cast was removed (Table 1-5).

There were 8 left and 6 right feet. 11 patients were male. All Beighton scores were 5 or less. All patients had increased lateral translation scores on the ATR side compared to the non-ruptured side($p<0.05$). 1 patient did not have an increased first ray instability score and was at 3 months from injury. All others had increased first ray instability and were at greater than 3 months from injury. All were low velocity injuries and none involved contact or significant sporting events.

No feet had clinical evidence of excess planovalgus upon visual inspection. There was no significant increase in the TA tightness. Data regarding this behaved non parametrically. 2 patients had lateral ankle radiographs. Formal radiographs were not requested for other patients. The Meary's axis in these 2 patients indicated cavus.

There was no significant difference in TA maximum AP thickness between affected and unaffected feet. Data for this was normally distributed. Pain score data was not normally distributed.

Title	Number of Patients
Male/Female ratio	11/3
Mean Age	54.57 range (37-79)
Mean foot length	26
Average time from rupture	345 days from injury (35-1378)
Number with tight TA (less than 10 degrees dorsiflexion)	7
Mean height cm / weight kg	167.48 cm/90.18 kg
Mean BMI	32.75
Mean time in POP	8 weeks

Table 1: Demographics of the patients.

	ATR foot (sd)	No ATR foot (sd)	P value
Mean lateral translation score	55.16mm (7.3)	25.69mm (9.3)	<0.05
Mean first ray instability score	9.04 mm (2.2)	3.62mm (2.95)	<0.05
Mean TA tightness	-2.29 degrees	0.29 degrees	>0.05
(degrees) (negative =dorsiflexion)			
Mean gastroc tightness (degrees) (negative =dorsiflexion)	-8 degrees	-7.64 degrees	>0.05
Inframalleolar pain (0-10)	2.07 (2.69)	0 (0)	<0.05
Retromalleolar pain (0-10)	3.00 (3.12)	0.13 (0.52)	<0.05
Valgus impingement pain (0-10)	2.27 (2.66)	0.13 (0.52)	<0.05
Thickness of the affected TA mm	11.1 (4.98)	8.0 (2.95)	>0.05

Table 2: Parameter comparison between ATR and non-ATR patients.

Mechanism of Injury	Number of Patients
Dorsiflexion	6
Plantarflexion/Tip Toe	6
Spontaneous	2

Table 3: Mechanism of injury of TA rupture.

No	Lateral Translation score(mm)	First Ray Instability score(mm)	TA tightness (degrees) -ve is dorsiflexion	Gastroc Tightness (degrees) -ve is dorsiflexion	Inframalleolar pain score	Retromalleolar pain score	Valgus Impingement score	Thickness of the affected TA (mm)
1	62	11.03	3	10	3	8	3	9.88
2	54.33	9.06	20	21	3	3	3	40
3	69.33	11.36	10	12	0	4	0	15.3
4	43.33	8.5	3	10	3	6	4	24.8
5	60.66	21	-20	-20	0	2	0	11
6	45	3.83	2	8	8	8	8	8.9
7	56.33	7.7	0	12	0	0	0	10.6
8	63.33	7.9	0	-13	0	5	0	72
9	51	1.76	12	18	3	0	3	12.6
10	58	11.1	6	6	0	0	0	10
11	46	11.6	4	2	2	0	3	14.4
12	54	10.7	10	18	0	2	0	11.9
13	55	8.3	22	22	7	7	9	5.8
14	54	0.9	11	20	0	0	4	12

Table 4: Parameters of the ATR foot.

No	Age	Gender	Height(cm)	Weight (Kg)	BMI	Affected Side	Mechanism of Injury	Time Since Injury (weeks)
1	42	F	162.6	59.3	22.4	Left	Dancing	13.9
2	79	M	175	78	25.47	Left	running	13
3	59	M	159	77.7	30.7	Left	run up a hill	53
4	62	M	177.5	119.6	38	Left	playing football	26.4
5	54	M	169.5	102.4	35.6	Right	Spontaneous	40.9
6	50	F	161	64.7	25	Right	jumping	5
7	30	M	174	79.2	26.2	Right	playing football	9.1
8	37	M	177.5	90.7	28.8	Right	playing football	45.1
9	51	F	169.2	74	25.8	Left	twisted movement	29.4
10	61	M	147.5	146	67.1	Left	spontaneous	33.9
11	53	M	173.9	92	30.4	Left	playing golf	29.6
12	74	M	182	118	35.6	Left	parking car	44.6
13	63	M	181	85.7	26.2	Right	playing football	198
14	49	M	135	75.3	41.3	Right	jumping	13.9

Table 5: Patient information.

Discussion

The Achilles Tendon (AT), the largest tendon in the human body, consists of fibers from the gastrocnemius and soleus muscles, with its architecture optimized for strength and functional efficiency. Capable of withstanding forces up to 9 kN or approximately 12.5 times body weight during activities like running, its peak stress is estimated at 70 MPa [15]. The peak age of rupture is 37.5 years [16].

Anatomy favours the function. AT fibre spiralisation increases strength and allows wide origin fibres to act at a single insertion, medially at the heel. This allows the soleus to function as a net inverter at heel rise. The crescent shaped insertion dissipates tendon stress [17].

Numerous theories attempt to explain the pathogenesis of ATR. The degenerative theory by Langergren, et al., hypothesised that ATRs are localised 2-6 cm proximal to the calcaneal insertion. A hypovascular area with low presence of intratendinous vessels, coupled with repeated trauma, prevents regeneration leading to rupture [18]. Histopathological analysis is corroborative demonstrating hypoxic and mucoid degeneration, poor vascular supply, tissue and cell necrosis, calcification, lipomatosis and irregular, degenerated collagen fibres in nearly all rupture sites [19-21]. The torsional trajectory theory assumes that the tendon compresses the transverse vincula. The functional over-pronation theory by Clement states that AT injury occurs due to a functional over-pronation and gastrocnemius soleus insufficiency. Incomplete synergism of agonist muscles, inefficient plantaris contraction or differences in muscle-tendon thickness quotients have been assumed to be contributory. Oblique loading, short initial length and maximal muscle contraction are thought contributory [22]. Tendon stiffness has also been implicated in ATRs. 5 minutes of passive stretching (preload) differentially decreases tendon stresses more in women (22.4%) compared to men (8.8%), possibly explaining greater ATR rates in men [23-25]. Differences in TA dorsiflexion were not present in our study (Table 2).

Pre-existing diseases such as rheumatoid arthritis, gout, ankylosing spondylitis, chronic uraemia, hyperparathyroidism only account for 2% of ATR. Barfred demonstrated complete ruptures occur in the absence of pre-existing etiological factors in healthy tendons [26].

The TA spans 3 joints including the knee, ankle and subtalar joint, passing medially to the subtalar joint axis to act as a net inverter [27,28]. The interaction between the subtalar joint and Achilles tendon has important mutual biomechanical implications. The subtalar axis' spatial location dynamically changes throughout the gait cycle [27]. Subtalar joint kinematics rather than heel varus or valgus position may play a greater role in ATR. Prior to toe off/heel rise the Center of Pressure [COP] line advances medially to the forefoot, the tibia externally rotates, externally rotating the talus and the subtalar axis spatial orientation, which is maximally lateralized distally towards the 4^{th} metatarsal or roughly 48% of the gait cycle [27]. The first ray thus generates a maximal net inversion forefoot moment (GRF) when transmitting 60% of weight in terminal stance. SL laxity defunctions this mechanism by allowing persistent medial talar head internal rotation, internally rotating the subtalar axis spatial orientation and changing the relationship of the medial calcaneal tuberosity and TA insertion to the subtalar axis [27].

Our study demonstrates significantly greater midfoot instability in terms of both Talonavicular laxity (TN) and first ray instability ($p<0.05$). Only 1 ATR foot had approximately equal first ray instability scores compared to the non-ruptured side and all ATR feet had greater TN laxity. This patient was about 3 months post ATR. Type 1 first ray instability occurs as a response to TN laxity. 2 patients had lateral radiographs demonstrating cavoid type feet.

8/14 had significant valgus impingement tenderness. Maximum tenderness was 8/10. Nine patients had coexisting retromalleolar tenderness. Both statically increased in ATR feet. Tenderness scores were between 2 and 8.

Our cohort of patients with TA ruptures didn't have gross valgus and morphologically looked relatively normal physiological valgus despite having medial arch laxity, thus allowing the potential development of an internally generated conflict force/moment that acts against an inversion at heel rise which is when the TA ruptures.

We postulate several prerequisites that biomechanically predisposes the TA to rupture, which we summarise here.

1. *Heel rise*

At heel rise there is acceleration and muscles including tibialis posterior and the TA are at maximum voluntary contraction (roughly 48% of gait cycle). Jumping or starting to sprint is a form of 'accelerated gait' requiring greater TA force. Body weight and TA contraction increases joint reaction forces/pressures across the subtalar and Tibio Talar (TT) joint, which increases downward talar head pressure. Maximum TA contraction acts at 10 times body weight combined with the force of the weight acting down exerts pressure on the SL via the talar head. The downward force applied by the anterior tibia on the anterior talar neck is opposed by an equal and opposite force exerted by navicular as an anterior buttress and inferomedially by the SL keeps the talus in static equilibrium thus allowing heel rise.

At heel strike the TA does not rupture as muscles are relatively inactive generating minimal force. The medial calcaneal tubercle (weight-bearing calcaneus) at heel strike lies medial to the subtalar joint axis creating an inversion Ground Reaction Force (GRF) moment around the subtalar axis that augments the theoretical line of action of the TA. SL laxity allows increased heel valgus that theoretically acts against any inversion TA pull. However, Soleus (62% cross sectional area) acts at only about 15% maximal amplitude [27,29] and is relatively inactive at this stage of gait, reflecting why TA does not rupture at heel strike. Studies evaluating ATR patients have demonstrated greater calcaneal inversion on heel strike [30], possibly reflecting the body's attempt to compensate for the midfoot laxity. Heel valgus / varus alone is therefore insufficient to explain if the TA would overload, with respect to the subtalar axis. A varus heel with SL laxity may still internally rotate the subtalar axis, therefore lateralise the forefoot GRF moment, thus generating an internal conflict moment.

2. *Laxity of the SL*

In our study all 14 patients had SL laxity or talonavicular axis laxity which allows greater non-physiological calcaneal eversion / valgus peaking in mid stance, at the point of increasing TA contraction. SL acts as a static restraint to heel valgus at the talonavicular axis. SL stability helps lock the midfoot and allows the stable first ray's inversion GRF moment to lie maximal away from the subtalar axis and augment hindfoot TA pull, thus allowing all moments act as a single composite inversion moment unit around the subtalar axis. SL laxity and FRI internally rotates the talus/subtalar axis permitting the TA to remain as a net inverter, whilst placing the forefoot as an everter, thus creating a moment of conflict. Progressive heel eversion due to increased SL strain puts the line of TA pull closer to the subtalar axis, diminishing its inverting moment for the same contraction force. Peak EMG soleus activity corresponds to heel raise, at just over 40% of the gait cycle, corresponding to when the TA commonly ruptures [7,32]. Increased EMG soleus activity demonstrated in Achilles tendinopathy representing sub-rupture overload pathology [31].

Eventually, excessive SL laxity increases heel valgus causing the calcaneal GRF and the TA to act lateral to the subtalar axis along with the forefoot GRF moment. As all act lateral to the subtalar axis thus there are no opposing moments at midstance or terminal stance from the latter two. The degree of SL laxity differentially changes the forefoot, hindfoot GRF and TA moments with respect to a progressively internally rotating subtalar axis. Furthermore, other factors such as foot length may play a role. Video analysis of TA ruptures demonstrate foot pronation (10 degrees) and external rotation consistent with deformities SL laxity produces [7,31].

3. *Internal Rotation of the Subtalar Axis*

A small degrees of SL strain amplifies subtalar axis arc of internal rotation of the at the forefoot, lateralising the forefoot GRF lateral to the subtalar axis, thus generating an eversion forefoot moment that opposes TA moment creating an internally generated zone of conflict. Evidence of subtalar axis internal rotation is seen in Achilles tendinopathy patients (representing sub-rupture TA overload) [33]. Reule demonstrated the subtalar joint's spatial orientation is internally rotated by 18 degrees in subjects with Achilles Tendinopathy [AT] an internal conflict moment compared to 11 degrees in unaffected subjects [33]. Munteanu and Barton demonstrated decreased peak tibial external rotation in AT patients by approximately 40% lower compared to control non-AT group [34]. They demonstrated that the group with previous Achilles tendinopathy exhibited an uncharacteristic internal rotation moment of the tibia present just after heel strike and just before toe-off. Excess internal tibial rotation arises from talus internal rotation due to SL laxity's failure to medially buttress the talus head. Williams showed 50% of Achilles tendinopathy patients demonstrated an internal rotation moment near heel strike and toe-off, correlating with our findings (Fig. 2,3) [35].

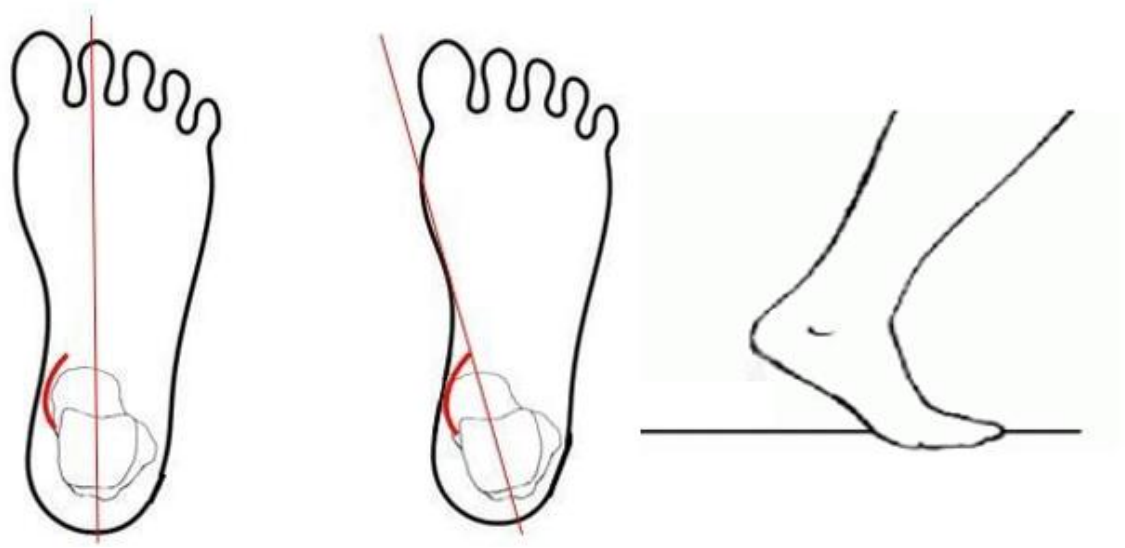

Figure 2: Small changes in talar spatial rotation changes the position of the subtalar axis in the forefoot significantly. (Approximately 9 degrees internal rotation for a 20 cm foot creates a 3 cm medial shift to the subtalar axis in the forefoot and loss of inversion moment). The third rocker puts greatest downward pressure on the talar head, with the TA maximally contracted.

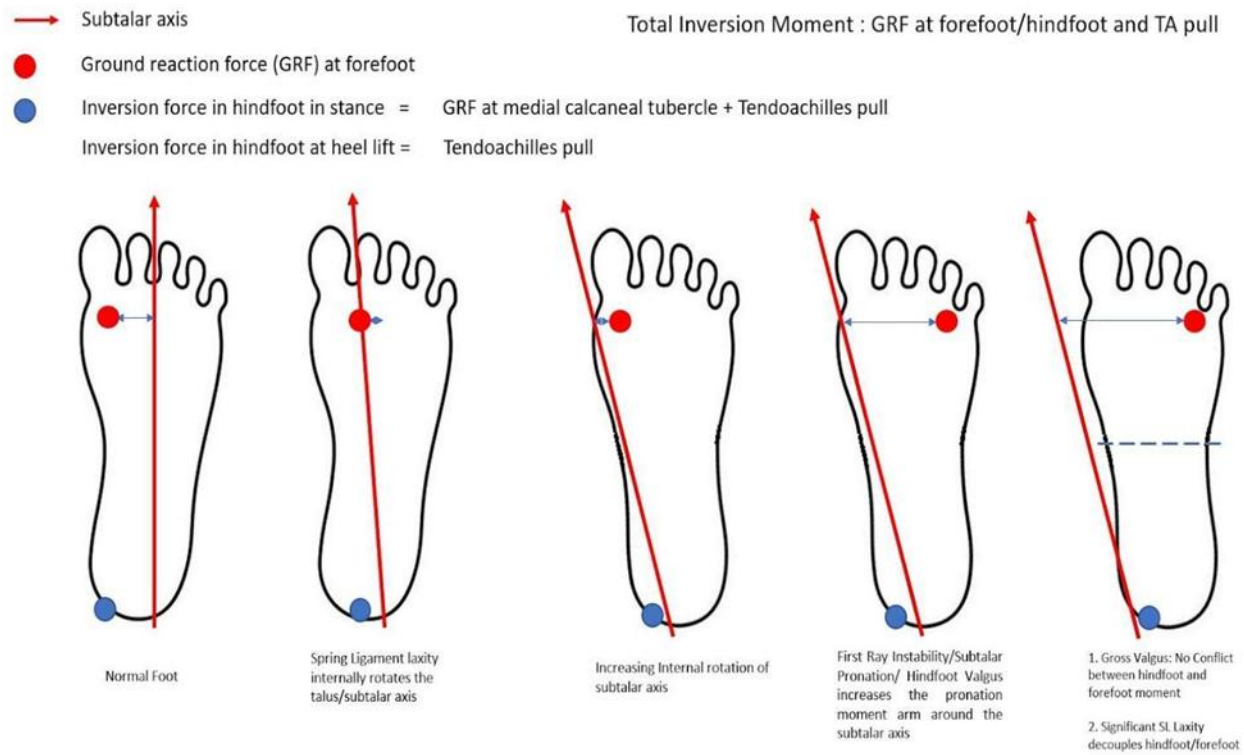

Figure 3: Total inversion Moment: GRF at forefoot/hindfoot and TA pull.

Medial Column Instability and Lateral Force Transfer

First ray is destabilised at around 3 months from SL laxity development [36]. 4 patients were identified at 3 months from the injury date who had POP and boot immobilisation. 3 out of 4 patients had increased FRI and only 1 patient had a stable first ray, but all had increased lateral translation scores. Pressure studies demonstrate a lateral shift in the forefoot load in ATR. These changes have been demonstrated in patients with Achilles tendinopathy also. Dong, et al., found a laterally shifted plantar pressure, 18-24 months after in feet with unilateral ATR. They found an increased subtalar eversion angle and decreased total

inversion [37]. Atik, et al., evaluated a single post Achilles rupture after 15 years follow up and demonstrated a lateral shift in the centre of pressure line [38]. Hahn and colleagues evaluated 13 patients using pedobarography and clinical assessment after flexor hallucis longus augmentation for TA ruptures (mean follow-up 46 months). A statistically increased forefoot pressure differences, force distribution and load transfer to the lateral foot were found when compared to the non-operated/injured leg. Whilst this was attributed to the FHL transfer, which diminishes phalanx power we believe unidentified SL laxity may have a role [39].

Coupling of Forefoot and Hindfoot Inverting Vectors

SL laxity decouples the forefoot and hindfoot moments. SL laxity differential changes the rate the forefoot and the hindfoot lose their inversion moment with respect to the subtalar axis. The maximum conflict arises when SL laxity causes persistent hindfoot/forefoot coupling with a maximum eversion moment at the forefoot with an inverting TA. None of our patients had gross valgus leading to no internal conflict moments. This explains why feet with gross valgus do not rupture, as the line of pull of the TA never enters the zone of conflict.

Susceptible Host

A sedentary host, despite having the above susceptible biomechanical profile may never rupture despite putting the TA into the 'zone of conflict' and therefore at risk. A host who plays basketball or sprints from still and with a maximally dorsiflexed foot, with no altered foot biomechanics may not generate an internal conflict moment to predispose the TA to rupture. The physiological strength of the collagen, BMI and level of activity would be additional factors determining SL laxity and rupture risk.

Peak ankle dorsiflexion and TA contraction exerts increased downward pressure on the talar neck. At peak dorsiflexion (40 degrees) the talar head hits the anterior part of the tibia, creating maximum downward pressure on the talar head and the SL, significantly exacerbated, during accelerated gait. A tight SL resists heel valgus at the midfoot rather and helps lock the midfoot and initiate a heel rise. The primary centre of rotation is at the metatarsal heads, but SL laxity creates a second centre of rotation in the midfoot, the primary one being at the metatarsal heads, creating a slight delay in dorsiflexion as confirmed by some studies. In our case 5 patients had a foot position described where the tibia was in the third rocker and the foot was about to generate a heel lift. This is consistent with Lemme, who showed that the foot was in dorsiflexion in 75% of cases [16].

Video evidence in basketball players demonstrates injury mechanisms [40]. The injured leg is loaded, with forward displacement of the centre of motion and an increase in hip and knee extension. There is an abrupt increase of ankle dorsiflexion, indicative of accelerated foot posture before forward and vertical propulsion. In one study, the ankle was abruptly dorsiflexed (29° at IF), externally rotated (15° at IF) and pronated (10° at IF) at the time of ATR. The measured maximum dorsiflexion angle of 40° was close to the range of physiological weight-bearing value. In addition, 48% demonstrated foot pronation. Just before take-off, when the foot is planted in dorsiflexion, the calf muscles eccentrically contract to prevent falling. 93% had no contact in their series of football injuries [7].

This is consistent with Lemme who evaluated 12 patients with video analysis and found peak rupture at toe off, consistent with peak acceleration when maximum thrust was about to be generated. Elastic energy stored in stretch then assists active contraction allowing the TA to generate an explosive propulsion force of 8 times body mass [40]. Tendon ruptures require greater force than this, intrinsically generated by an opposite GRF acting pathologically lateral to the subtalar axis. Intrinsic TA fibre arrangement may also play a role. More internal rotation of the TA fibres may rupture less as a greater inversion moment is generated. This has been insufficiently examined.

TA tightness may lead to an early heel rise and prolonged stance phase putting downward pressure on the talar head and therefore increase the duration of biomechanical risk profile for TA rupture. In our series there was no excessively tight TA. This was not found as a confounding factor in our cohort. Furthermore, TA thickness was not found to be significant in our study. TA thickness varies in its presentation depending on stage. Majewski, et al., demonstrated TA thickness peaked at 24 months regardless of whether treated percutaneously, open or non-operatively (17 mm) after an ATR. Initial thickness would increase due to hematoma, followed by organisation. Mature tendons then remodel to become relatively normal size (9-11mm). This is different to tendinopathic tendons which have a larger size [41].

Theoretically, medial column instability may have developed after the injury because of loss of the net inverting vector of the TA that may off load ligament stress. However, patient 14 is important to note, who was examined after their period of POP immobilisation and had not progressed onto loading the foot. This patient demonstrated medial column instability prior to mechanical loading. Patient 1 and 2 also finished their POP immobilisation time and were in their boot/just out of their boot. Both had evidence of medial column instability. Previous studies have implied first ray instability occurs approximately 3-6 months after injury to the SL [36].

Limitations

We accept that this study does not establish a temporal relationship between medial column instability onset and TA rupture. Formally algometry threshold testing was not performed in relation to the tibialis posterior tenderness.

Conclusion

Our study demonstrates increased medial column laxity is present in all ATR feet. The presence of signs of TP overload in some feet further suggests a common pathogenesis. Intrinsic foot biomechanics as a risk factor that predisposes the foot to ATR has not been described to date and helps rationally explain how huge intrinsic non-contact forces can be generated to overload the TA. We advocate using a medial arch support to help restore the subtalar axis and offload the midfoot to offload the TA. Midfoot laxity should be assessed in TA ruptures, to help restore biomechanics and protect the foot from future re-ruptures.

Conflict of Interests

The authors declare that they have no conflict of interest in this paper.

Acknowledgments

Not applicable

Funding/Sponsorship

Not applicable

References

1. Kim GK. The risk of fluoroquinolone-induced tendinopathy and tendon rupture: what does the clinician need to know? J Clinical and Aesthetic Dermatology. 2010;3(4):49.
2. Maffulli N, Waterston SW, Squair J, Reaper J, Douglas S. Changing incidence of Achilles tendon rupture in Scotland: a 15-year study. Clinical J Sport Medicine. 1999;9(3):157-60.
3. Houshian S, Tscherning T, Riegels-Nielsen P. The epidemiology of Achilles tendon rupture in a Danish county. Injury. 1998;29(9):651-4.
4. Suchak AA, Bostick G, Reid D, Blitz S, Jomha N. The incidence of Achilles tendon ruptures in Edmonton, Canada. Foot and ankle international. 2005;26(11):932-6.
5. Möller A, Åström M, Westlin NE. Increasing incidence of Achilles tendon rupture. Acta Orthopaedica Scandinavica. 1996;67(5):479-81.
6. Leppilahti J, Puranen J, Orava S. Incidence of Achilles tendon rupture. Acta Orthopaedica Scandinavica. 1996;67(3):277-9.
7. Della Villa F, Buckthorpe M, Tosarelli F, Zago M, Zaffagnini S, Grassi A. Video analysis of Achilles tendon rupture in male professional football (soccer) players: injury mechanisms, patterns and biomechanics. BMJ Open Sport and Exercise Medicine. 2022;8(3):e001419.
8. Amin NH, McCullough KC, Mills GL, Jones MH, Cerynik DL, Rosneck J, et al. The impact and functional outcomes of Achilles tendon pathology in National Basketball Association players. Clinical RESEARCH on Foot and Ankle. 2016;4(3).
9. Johns W, Walley KC, Seedat R, Thordarson DB, Jackson B, Gonzalez T. Career outlook and performance of professional athletes after Achilles tendon rupture: a systematic review. Foot and Ankle International. 2021;42(4):495-509.
10. Noback PC, Jang ES, Cuellar DO, Seetharaman M, Malagoli E, Greisberg JK, et al. Risk factors for achilles tendon rupture: a matched case control study. Injury. 2017 ;48(10):2342-7.
11. Partridge L, Rajbhandari S. Achilles tendon in diabetes. Current diabetes reviews. 2017;13(4):424-7.
12. De Jonge S, Rozenberg R, Vieyra B, Stam HJ, Aanstoot HJ, Weinans H, et al. Achilles tendons in people with type 2 diabetes

show mildly compromised structure: an ultrasound tissue characterisation study. British J Sports Medicine. 2015;49(15):995-9.
13. Pasapula C, Devany A, Magan A, Memarzadeh A, Pasters V, Shariff S. Neutral heel lateral push test: The first clinical examination of spring ligament integrity. The Foot. 2015;25(2):69-74.
14. Yao L, Gentili A, Cracchiolo A. MR imaging findings in spring ligament insufficiency. Skeletal Radiology. 1999;28:245-50.
15. Komi PV, Fukashiro S, Järvinen M. Biomechanical loading of Achilles tendon during normal locomotion. Clinics in Sports Medicine. 1992;11(3):521-31.
16. Lemme NJ, Li NY, DeFroda SF, Kleiner J, Owens BD. Epidemiology of Achilles tendon ruptures in the United States: athletic and nonathletic injuries from 2012 to 2016. Orthopaedic J Sports Medicine. 2018;6(11):2325967118808238.
17. Lohrer H, Arentz S, Nauck T, Dorn-Lange NV, Konerding MA. The Achilles tendon insertion is crescent-shaped: an in vitro anatomic investigation. Clinical Orthopaedics and Related Research®. 2008;466(9):2230-7.
18. Langergren C, Lindholm A. Vascular distribution in the Achilles tendon. Acta Chir Scand. 1958;116:491.
19. Arner O. Subcutaneous rupture of the Achilles tendon. A study of 92 cases. Acta Chir Scand Suppl. 1959;116(239):1-51.
20. Jozsa L, Kannus P. Histopathological findings in spontaneous tendon ruptures. Scandinavian J Medicine and Science in Sports. 1997;7(2):113-8.
21. Fox JM, Blazina ME, Jobe FW, Kerlan RK, Carter VS, Shields Jr CL, et al. Degeneration and rupture of the Achilles tendon. Clinical Orthopaedics and Related Research®. 1975;107:221-4.
22. Clement DB, Taunton JE, Smart GW. Achilles tendinitis and peritendinitis: etiology and treatment. The Am J Sports Medicine. 1984;12(3):179-84.
23. Morse CI, Degens H, Seynnes OR, Maganaris CN, Jones DA. The acute effect of stretching on the passive stiffness of the human gastrocnemius muscle tendon unit. The J Physiology. 2008;586(1):97-106.
24. Nakamura M, Ikezoe T, Takeno Y, Ichihashi N. Acute and prolonged effect of static stretching on the passive stiffness of the human gastrocnemius muscle tendon unit in vivo. J Orthopaedic Research. 2011;29(11):1759-63.
25. Intziegianni K, Cassel M, Hain G, Mayer F. Gender differences of Achilles tendon cross-sectional area during loading. Sports Medicine Int Open. 2017;1(04):E135-40.
26. Barfred T. Experimental rupture of the Achilles tendon: comparison of various types of experimental rupture in rats. Acta Orthopaedica Scandinavica. 1971;42(6):528-43.
27. Brockett CL, Chapman GJ. Biomechanics of the ankle. Orthopaedics and Trauma. 2016;30(3):232-8.
28. Lewis GS, Kirby KA, Piazza SJ. Determination of subtalar joint axis location by restriction of talocrural joint motion. Gait and Posture. 2007;25(1):63-9.
29. Hansen P, Kovanen V, Hölmich P, Krogsgaard M, Hansson P, Dahl M, et al. Micromechanical properties and collagen composition of ruptured human achilles tendon. The Am J Sports Medicine. 2013;41(2):437-43.
30. Sun D, Fekete G, Baker JS, Mei Q, István B, Zhang Y, Gu Y. A pilot study of musculoskeletal abnormalities in patients in recovery from a unilateral rupture-repaired Achilles tendon. Int J Environmental Research and Public Health. 2020;17(13):4642.
31. McHugh MP, Orishimo KF, Kremenic IJ, Adelman J, Nicholas SJ. Electromyographic evidence of excessive Achilles tendon elongation during isometric contractions after Achilles tendon repair. Orthopaedic J Sports Medicine. 2019;7(11):2325967119883357.
32. Hoenig T, Gronwald T, Hollander K, Klein C, Frosch KH, Ueblacker P, Rolvien T. Video analysis of Achilles tendon ruptures in professional male football (soccer) reveals underlying injury patterns and provides strategies for injury prevention. Knee Surgery, Sports Traumatology, Arthroscopy. 2023;31(6):2236-45.
33. Reule CA, Alt WW, Lohrer H, Hochwald H. Spatial orientation of the subtalar joint axis is different in subjects with and without Achilles tendon disorders. British J Sports Medicine. 2011;45(13):1029-34.
34. Munteanu SE, Barton CJ. Lower limb biomechanics during running in individuals with achilles tendinopathy: a systematic review. J Foot and Ankle Research. 2011;4:1-7.
35. Williams III DB, Zambardino JA, Banning VA. Transverse-plane mechanics at the knee and tibia in runners with and without a history of achilles tendonopathy. J Orthopaedic and Sports Physical Therapy. 2008;38(12):761-7.
36. Pasapula C, Ali AM, Kiliyanpilakkil B, Hardcastle A, Koundu M, Gharooni AA, et al. High incidence of spring ligament laxity in ankle fractures with complete deltoid ruptures and secondary first ray instability. The Foot. 2021;46:101720.
37. Sun D, Fekete G, Mei Q, Gu Y. Gait abnormality and asymmetry analysis after 18–24 months surgical repair of unilateral

Achilles tendon rupture. J Medical Imaging and Health Informatics. 2019;9(3):552-60.
38. Atik OŞ, Bölükbaşı S, Kanatlı U. Does surgical repair of acute Achilles tendon rupture cause structural changes on 15-year follow-up? Joint Diseases and Related Surgery. 2011;22(1):054-5.
39. Hahn F, Maiwald C, Horstmann T, Vienne P. Changes in plantar pressure distribution after Achilles tendon augmentation with flexor hallucis longus transfer. Clinical Biomechanics. 2008;23(1):109-16.
40. Lemme NJ, Li NY, Kleiner JE, Tan S, DeFroda SF, Owens BD. Epidemiology and video analysis of Achilles tendon ruptures in the National Basketball Association. The American Journal of Sports Medicine. 2019;47(10):2360-6.
41. Majewski M, Lehmann M, Dick W, Steinbrück K. Value of sonography to monitor the course of Achilles tendon rupture after treatment-comparison of conservative therapy, percutaneous tendon adaptation, and open suture. Der Unfallchirurg. 2003;106:556-60.